

Cardiovascular adaptation after liver transplantation. Ventricular changes detect by 2d echocardiography and by speckle tracking

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Background. Patients who underwent liver transplantation (LT) may suffer from heart disease that can be related to the liver disease itself or to other associated pathologies. It has been suggested that there is a specific heart disease associated with cirrhosis, termed cirrhotic cardiomyopathy which is characterized by the presence of increased baseline cardiac output, systolic and diastolic left ventricular (LV) dysfunction and increased in pulmonary artery systolic pressure (PASP). The aim of the study was to evaluate the cardiac structural and functional changes after LT and eventually to determine the improvement in PASP.

Method. 51 patients were considered for the analysis who had a good quality pre and post LT echocardiograms. The echo-study was done preLT and repeated within 4 months and 3 years after LT. All studies were red of-line, global longitudinal strains (GLS) of the LV and right ventricle (RV) were analyzed using TOMTEC application. A Paired T-test was used to compare the echocardiographic parameters. The group was also divided according to tertiles of pre LT PASP for the evaluation of pulmonary pressure.

Results . Patients` mean age was 58.1 ± 7.8 years, 32 (62.6%) men, mean time between the 2 echocardiographic studies was 529.2 ± 471 days. After LT all the patients were on immunosuppressant therapy (calcineurin inhibitors, ciclosporin and/or tacrolimus). After LT blood pressure (BP) (83.4 ± 16 vs 91.5 ± 14 mmHg, $p = 0.009$ for mean BP), heart rate (72.5 ± 15.2 vs 80.6 ± 15.3 bpm, $p = 0.004$), increased as LV mass index (78.6 ± 21.1 to 91.4 ± 29 , $p = 0.003$) and relative wall thickness (0.34 ± 0.06 to 0.39 ± 0.08 , $p = 0.001$). LV ejection fraction did not change while there was a significant decrease in LV GLS ($-20.9 \pm 4.4\%$ vs $-17.4 \pm 3.9\%$, $p < 0.0001$), impaired diastolic function (E/A 1.12 ± 0.5 vs 0.94 ± 0.4 , $p = 0.002$) and increase in LV diastolic filling pressure (E/E' 7.7 ± 3.6 vs 8.9 ± 3.6 , $p = 0.018$). PASP increased (26.6 ± 8 vs 30.8 ± 11 mmHg, $p = 0.018$) and TAPSE (24.1 ± 4.5 vs 21.6 ± 3.9 mm, $p = 0.002$) decreased. The pre and post echo data, were divided in preLT PASP to see if there was any tendency to decrease in PASP after LT. A progressive increase in LV remodeling and impaired diastolic function, RV- pulmonary arterial coupling decreased as an index of RV maladaptation to the increase PASP as tertiles of in PASP increased.

Conclusion. Increased in BP has been found in patients after LT , likely related to immensuppressive therapy. LV remodeling, impaired diastolic function were likely a consequence of increase in BP and the increased in PASP and worst RV- pulmonary circulation coupling was secondary to impaired diastolic function and increased filling pressure. After LT, patients require particular clinical attention and echocardiographic monitoring included GLS for target organ damage and prompt adequate therapy at least in terms of BP control to avoid later cardiovascular complications.